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Original article

Association between C-reactive protein level and echocardiography assessed left ventricular function in first ST-segment elevation myocardial infarction patients who underwent primary coronary intervention☆

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ABSTRACT

Background: An elevated C-reactive protein (CRP) level is associated with adverse outcomes in patients with acute myocardial infarction (AMI). Although CRP levels have been shown to be associated with left ventricular (LV) systolic function and remodeling in AMI, little is known about their relation to early LV diastolic function.

Methods: We retrospectively studied 173 consecutive patients <75 years of age with first ST-segment elevation MI (STEMI) that was treated by primary percutaneous coronary intervention (PPCI). They had presented within 24 h of chest pain onset and their CRP levels were determined within 6 h of hospital admission. They all underwent echocardiography within 3 days of admission and were stratified by CRP tertiles.

Results: The cut-off points for the CRP tertiles were <2.6 mg/L, 2.6–7.9 mg/L, and >7.9 mg/L. Patients with higher CRP levels had a significantly higher mean mitral inflow E wave velocity (68 ± 16 cm/s vs 77 ± 19 cm/s vs 76 ± 17 cm/s; $p = 0.02$), a higher E/average e' (8.9 ± 1.9 vs 9.8 ± 2.8 vs 10.4 ± 3.2; $p = 0.02$), and a higher systolic pulmonary artery pressure (27 ± 6 mmHg vs 30 ± 8 mmHg vs 32 ± 10 mmHg; $p = 0.04$). Elevated CRP levels were associated with more advanced diastolic dysfunction than normal CRP levels ($p = 0.04$). The admission CRP level was an independent predictor of average E/e' ratio (multivariate analysis).

Conclusion: Admission CRP levels are associated with echocardiographic parameters of elevated LV filling pressure in patients with STEMI treated with PPCI.

Introduction

Ischemic injury and myocardial necrosis following an acute myocardial infarction (AMI) incite an acute inflammatory response. Of the various proinflammatory cytokines, C-reactive protein (CRP) has emerged as a powerful and independent predictor of heart failure (HF) and long-term mortality [1–3]. Elevation of CRP levels in AMI has been shown to correlate with reduced left ventricular (LV) systolic function [2,3] and long-term LV remodeling [4,5]. While an association between CRP and LV diastolic dysfunction has been previously reported in patients with chronic coronary artery disease [6,7], little is known about its relation to early diastolic LV function in the setting of an AMI.

The presence of echocardiographic parameters of diastolic dysfunction following an AMI, especially those suggestive of elevated LV filling pressures, are associated with adverse remodeling, increased incidence of HF, and worse survival [8–11]. We evaluated the relation between CRP levels on admission and diastolic LV function in patients presenting with first ST-segment elevation MI (STEMI) who underwent successful reperfusion with primary percutaneous coronary intervention (PPCI). We hypothesized that higher admission CRP levels would be associated with poorer diastolic LV function.

☆ All authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

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Methods and patients

We retrospectively studied 258 consecutive patients with an acute STEMI who had been admitted to the cardiac intensive care unit (CICU) of the Tel-Aviv Sourasky Medical Center between May 2011 and January 2013, and treated with PPCI. The baseline demographic, clinical, echocardiographic, and angiographic features test results were retrieved from medical files and the hospital database. A total of 237 patients had serum CRP levels obtained immediately following primary PCI. Excluded were 43 patients older than 75 years (in order to avoid age-related diastolic dysfunction) [12], 15 patients with a previous MI, and 3 patients who had severe hemodynamic instability (e.g. cardiogenic shock at admission defined as persistent systolic blood pressure <90 mmHg and nonresponsive to fluid replacement, or the need for inotropes or intra-aortic balloon pumping to maintain blood pressure ≥90 mmHg). Another 3 patients with a known alternative reason for CRP elevation (2 with recent infection and 1 with connective tissue disease) were excluded, leaving a final study population of 173 patients.

The diagnosis of STEMI in each patient was defined by a typical history of chest pain, diagnostic electrocardiographic changes, and serial elevation of serum cardiac biomarkers [13]. The electrocardiographic criterion for the diagnosis of STEMI was an ST-segment elevation of ≥1 mm in >2 adjacent leads. Symptom duration was defined as the time from symptom onset (usually chest pain or discomfort) to emergency room (ER) admission. The patients were treated according to the discretion of the senior attending physician in the CICU. PPCI was performed within 90 min of hospital admission. Blood samples for CRP were drawn in all 173 patients within 6 h of admission to the emergency room. The complete blood count parameters were measured using a Coulter STKS electronic counter (Beckman–Coulter, Hialeah, FL, USA). Wide-range CRP analysis was performed by the Bayer wide-range assay as previously described [14].

All patients underwent a screening echocardiographic examination within 6-72 h of CICU admission. Echocardiography was performed by Philips IE-33, GE, and Vivid 3 models equipped with S5-1 transducers (Philips Healthcare, Andover, MA, USA). LV diameters and interventricular septal and posterior wall width were measured from the parasternal short axis by means of a 2-dimensional (2D) or a 2D-guided M-mode echocardiogram of the LV at the papillary muscle level using the parasternal short-axis view [15]. LV ejection fraction (LVEF) was calculated by the Quinones method. The 16-segment model was used for scoring the severity of segmental wall motion abnormalities according to the American Society of Echocardiography [16]. Early transmitral flow velocity (E), deceleration time, and early diastolic mitral annular velocity (e') were measured in the apical 4-chamber view to provide an estimate of LV diastolic function [17]. The ratio of peak E to peak septal, lateral, and average e' (mitral E/e' ratio) was calculated from the average of at least 3 cardiac cycles. Left atrial volume was calculated using the biplane area length method at end systole [18]. Cardiac output was calculated as the product of aortic stroke volume and heart rate as demonstrated on pulse wave Doppler. Valvular regurgitation was qualitatively assessed using color Doppler according to the guidelines of the American Society of Echocardiography (normal/trivial = 1, mild = 2, moderate = 3, severe = 4) [19]. Diastolic function was assessed by integrating measurements of the mitral inflow, left atrial volume, and Doppler tissue imaging of the mitral annulus using the average annulus velocity, and classified into four categories: normal diastolic function = 0, impaired relaxation = 1, pseudo-normal = 2 and restrictive pattern = 3, based on recent guidelines [17]. Right atrial (RA) pressure was estimated by the inferior vena cava diameter as well as its response to inspiration as previously described [20]. Briefly, expiratory and inspiratory inferior vena cava (IVC) diameters and percent collapse were measured in subcostal views within 2 cm of the right atrium. IVC diameter <2.1 cm that collapsed >50% with a sniff suggested a normal RA pressure (assigned as 5 mmHg), whereas an IVC diameter >2.1 cm that collapsed <50% with a sniff suggested a high RA pressure (15 mmHg). In patients with IVC diameter <2.1 cm and no collapse (<20%) with a sniff, RA pressure was upgraded to 20 mmHg. In indeterminate cases in which the IVC diameter and collapse did not fit this paradigm, secondary indices of elevated RA pressure were integrated. If uncertainty remained, RA pressure was left as intermediate value of 10 mmHg. In 122 patients with measurable tricuspid regurgitation jet on Doppler echocardiography peak systolic pulmonary artery pressure (SPAP) was estimated using the modified Bernoulli formula $(4 \times TRV^2_{max}) + RAP$, where TRV_{max} is the peak systolic tricuspid regurgitation velocity at end expiration, and RAP is the right atrial pressure. SPAP was unmeasurable in the remaining 51 patients.

LV mass index was calculated by Devereux's formula [15] considering the diastolic measurements of left ventricular internal diameter interventricular septal thickness and posterior wall thickness. Relative wall thickness (RWT) was calculated as: 2 × posterior wall thickness divided by LV diastolic diameter [15].

All data were summarized and displayed as mean (± standard deviation) for continuous variables and as number (percentage) of patients in each group for categorical variables. Patient groups were stratified according to CRP tertiles. Since CRP displayed irregular distributions, we used logarithmic transformation which converted the distributions to normal ones for all statistical procedures. Therefore, all results of CRP are expressed as back-transformed geometrical means. The p-values were than calculated using the ANOVA and the Pearson chi-square tests for the continuous and categorical variables, respectively. Linear regression models were performed at the ENTER mode, where E/e' was defined as the dependant variable and adjusted to age, gender, LVEF, peak troponin, ischemia duration (defined as time from symptom onset to reperfusion), anterior MI, coronary artery disease severity, as well as CRP. All of the analyses were considered significant at a 2-tailed p-value <0.05. The SPSS statistical package was used to perform all statistical evaluations (SSPS, Chicago, IL, USA).

Results

The study cohort included a total of 173 consecutive patients (mean age 57 ± 9 years, range 34–75, 82% males). CRP levels were measured at a median of 6.1 h after symptom onset (25th–75th percentile 5–11 h). The median CRP value was 4.7 mg/L (25th–75th percentile 1.84–10.87 mg/L). Baseline echocardiograms were obtained at 1.6 ± 1.4 days following admission. Table 1 presents the clinical, laboratory, and angiographic data of patients according to CRP tertiles. The cut-off points for the CRP tertiles were <2.6 mg/L, 2.6–7.9 mg/L, and >7.9 mg/L. Patients with higher CRP levels had longer symptom duration prior to ER admission and more severe coronary artery disease. Table 2 displays the echocardiographic parameters in groups stratified by CRP tertiles. Patients with higher CRP levels had a significantly higher mean mitral inflow E wave velocity ($p = 0.02$), a higher septal E/e' ($p = 0.02$), a higher lateral E/e' ($p = 0.02$), higher E/average e' ($p = 0.02$) ratios, higher prevalence of pathologic E/e' ratio (E/e' lateral >12 and E/e' septal >15), and a higher SPAP ($p = 0.04$). There were no significant changes in LVEF, cardiac output, left atrial volume, and other echocardiographic parameters between the groups. No significant change in CRP levels was found between those with left atrial volume index ≤ and >32 (9.6 ± 14.9 vs 11.8 ± 9.2; $p = 0.42$).

Table 1
Clinical characteristics, angiographic findings and laboratory biomarkers by C-reactive protein (CRP) tertiles.

Variable	Tertile 1 <2.6 mg/L, $n=58$	Tertile 2 2.6–7.9 mg/L, $n=59$	Tertile 3 >7.9 mg/L, $n=56$	p-Value*
Age, years (mean ± SD)	57 ± 9	56 ± 9	56 ± 9	NS
Male	50 (86%)	48 (82%)	46 (82%)	NS
Hypertension	21 (36%)	23 (39%)	17 (30%)	NS
Hyperlipidemia	32 (55%)	20 (34%)	20 (36%)	0.04
Smoker	28 (48%)	43 (73%)	36(64%)	0.02
Diabetes mellitus	8 (14%)	7 (12%)	9 (16%)	NS
Family history	13 (22%)	15 (25%)	13 (23%)	NS
Statin treatment	22(38%)	13(22%)	15 (26%)	NS
Renin angiotensin system blockers	14 (23%)	17 (28%)	14 (25%)	NS
Anterior infarct location	34 (59%)	26 (44%)	22 (39%)	NS
Right ventricular infarction	1 (1%)	2 (3%)	4 (7%)	NS
Symptom duration (min)	150 (112)	200 (525)	225 (493)	0.007
Median (interquartile range)				
Number of narrowed coronary arteries				
1	34 (59%)	27 (46%)	20 (36%)	0.03
2	19 (32%)	17 (29%)	19 (34%)	0.02
3	5 (9%)	15 (25%)	17 (30%)	0.002
Proximal infarct-related artery narrowing	31 (53%)	31 (53%)	29 (52%)	NS
Peak troponin ng/mL (mean ± SD)	44 ± 8	62 ± 11	37 ± 7	NS
Peak CPK, IU/l (mean ± SD)	1588 ± 205	1546 ± 204	1749 ± 233	NS
CRP, mg/L (mean ± SD)	1.31 ± 0.74	4.97 ± 1.46	26.1 ± 22.4	<0.001
Fibrinogen, mg/dL (mean ± SD)	263 ± 54	310 ± 59	366 ± 88	<0.001
White blood cell,$10^3/\mu L$ (mean ± SD)	11.4 ± 3.0	12.1 ± 3.5	11.4 ± 3.0	NS
Neutrophil count, $10^3/\mu L$ (mean ± SD)	7.6 ± 2.9	8.2 ± 3.4	8.3 ± 2.6	NS

CPK, creatine phosphokinase.
* The p-values were calculated using the ANOVA and the Pearson chi-square tests for the continuous and categorical variables, respectively. NS indicates two-tailed non-significance ($p > 0.05$).

Patients with higher CRP levels also had a more advanced diastolic dysfunction grade ($p = 0.04$; Fig. 1). Admission serum CRP levels significantly correlated with the mitral E/average e' ratio ($p < 0.001$; Fig. 2). CRP emerged as an independent predictor of E/average e' ratio in the multivariate analysis model (Table 3).

Discussion

This is the largest echocardiographic evaluation of the relation between CRP levels and LV function in patients with acute STEMI treated by PPCI. Our main finding is that even a mild elevation of the CRP level (>2.6 mg/L) in these STEMI patients was

Table 2
Echocardiographic characteristics by C-reactive protein (CRP) tertiles.

Variable	Tertile 1 <2.6 mg/L, $n=58$	Tertile 2 2.6–7.9 mg/L, $n=59$	Tertile 3 >7.9 mg/L, $n=56$	p-Value*
Biplane LV ejection fraction	48 ± 8	48 ± 8	46 ± 8	NS
Wall motion index	1.54 ± 0.4	1.53 ± 0.4	1.61 ± 0.3	NS
Heart rate (beats/min)	74 ± 10	73 ± 11	75 ± 13	NS
Systolic blood pressure (mmHg)	135 ± 18	139 ± 19	136 ± 20	NS
Diastolic blood pressure (mmHg)	81 ± 11	81 ± 14	81 ± 12	NS
Cardiac output (L/min)	5.1 ± 1.2	5.1 ± 1.2	5.2 ± 1.1	NS
Left atrial volume (ml^3)	62 ± 16	60 ± 16	61 ± 17	NS
Left atrial volume index (ml/m^2)	32 ± 8	31 ± 8	32 ± 8	NS
Mitral inflow E wave (cm/s)	68 ± 16	77 ± 19	76 ± 17	0.02
Mitral inflow A wave (cm/s)	65 ± 22	70 ± 21	72 ± 21	NS
Mitral inflow E/A ratio	1.15 ± 0.45	1.19 ± 0.45	1.16 ± 0.46	NS
Septal e' (cm/s)	6.9 ± 2	6.8 ± 1.9	6.6 ± 1.8	NS
E wave velocity/septal e′	10.3 ± 2.7	12.3 ± 5.6	12.2 ± 3.7	0.02
Lateral e' (cm/s)	9.2 ± 2.6	9.3 ± 2.7	8.5 ± 2.6	NS
E wave velocity/lateral e'	7.9 ± 2.3	8.7 ± 2.6	9.5 ± 3.3	0.02
E wave velocity/average e'	8.9 ± 1.9	9.8 ± 2.8	10.4 ± 3.2	0.02
Mitral E deceleration time (ms)	181 ± 46	179 ± 47	176 ± 38	NS
LV end diastolic dimension (mm)	48 ± 7	45 ± 13	49 ± 8	NS
LV end systolic dimension (mm)	31 ± 7	29 ± 9	32 ± 7	NS
Septal thickness (mm)	11 ± 2	11 ± 2	12 ± 2	NS
Posterior wall thickness (mm)	10 ± 2	9 ± 3	10 ± 2	NS
Relative wall thickness	0.43 ± 0.07	0.43 ± 0.08	0.41 ± 0.07	NS
LV mass index (g/m^2)	101 ± 30	101 ± 37	98 ± 29	NS
SPAP (mmHg)	27 ± 6	30 ± 8	32 ± 10	0.04
Right atrial pressure (mmHg)	5 ± 2	6 ± 3	6 ± 3	NS
Moderate/severe MR, n (%)	1 (1.7%)	2 (3.3%)	2 (3.5%)	NS
E/septal $e' \geq 15$, n (%)	1 (1.7%)	10 (17%)	11 (20%)	0.001
E/Lateral $e' \geq 12$, n (%)	2 (3.4%)	7 (11%)	10 (18%)	0.04

Data are expressed as mean ± SD. LV, left ventricle; SPAP, systolic pulmonary artery pressure; MR, mitral regurgitation.
* The p-values were calculated using the ANOVA and the Pearson chi-square tests for the continuous and categorical variables, respectively. NS indicates two-tailed non-significance ($p > 0.05$).

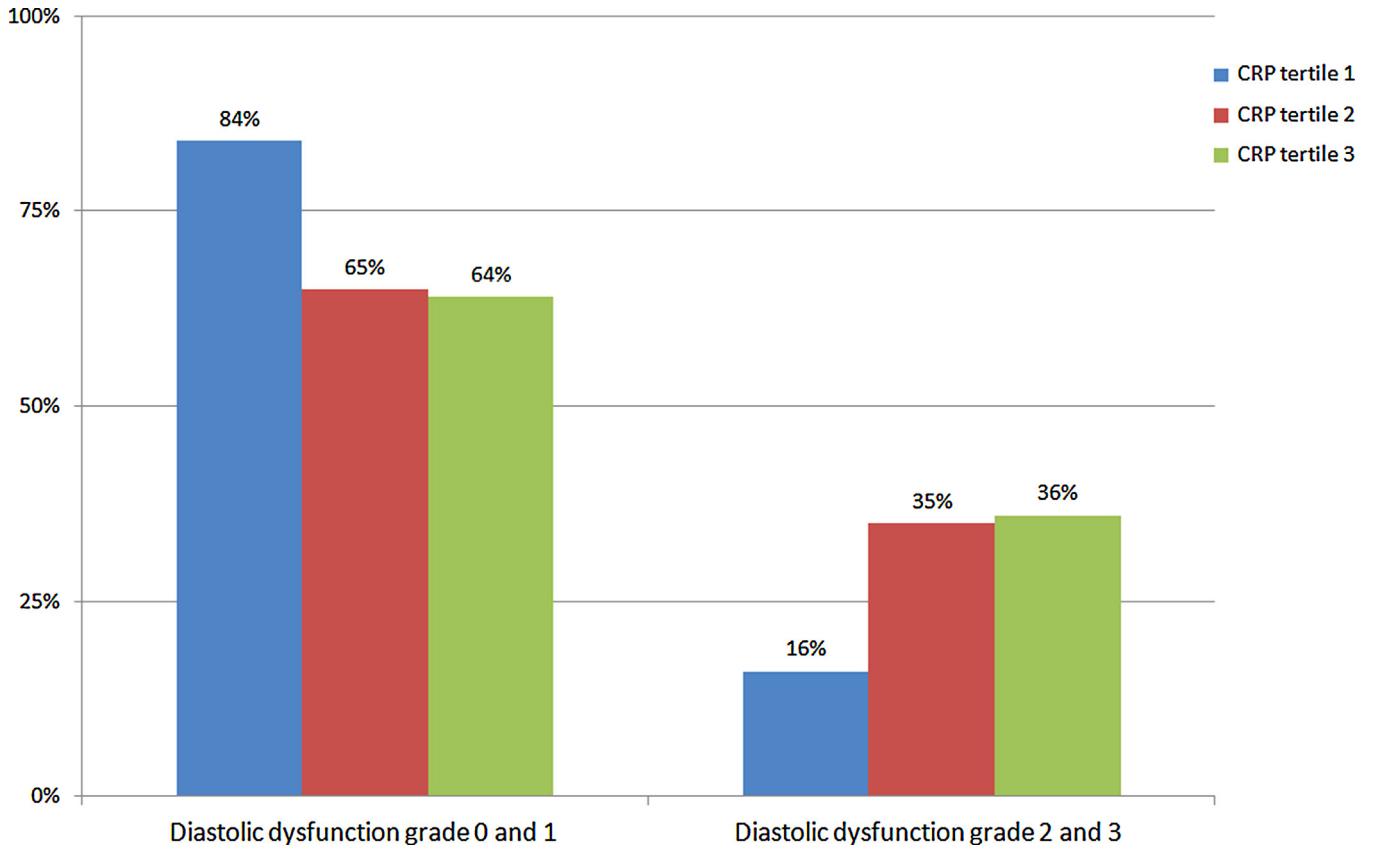

Fig. 1. Correlation between admission C-reactive protein (CRP) tertiles and diastolic function grading ($p = 0.04$).

associated with worsening of diastolic function and elevated LV filling pressure, independent of LV systolic function or cardiac output. Previous studies have demonstrated the relation of early CRP elevation with LV systolic function, future HF, and mortality in AMI patients [1–5]. Suleiman et al. demonstrated a graded association between CRP levels obtained within 24 h of symptom onset, worsening in LV systolic function, and future HF and mortality [3]. In that study, however, changes in LV systolic function as well as future HF and mortality were present among patients with moderate or severe CRP levels (>13.5 mg/dL). In addition, fewer than half of their patients received any reperfusion therapy (thrombolysis or PPCI). Arruda-Olson et al. reported that CRP elevation at the initial phase of an AMI was associated with worse diastolic dysfunction as well as worse functional mitral regurgitation, which was independent of LV systolic function [21]. In that report, however, other echocardiographic diastolic parameters, such as mitral valve diastolic flow velocities, mitral annular velocities, SPAP, and left atrial volume were not evaluated. Moreover, almost half of their patients who had CRP levels >10 mg/L had an infection within 2 weeks prior to their index hospitalization with an MI [21], thus obscuring a direct relation between CRP to diastolic dysfunction following the occurrence of an MI.

There are now convincing data that CRP is an independent predictor of future cardiovascular events, including cardiovascular death, MI, stroke, revascularization, the development of peripheral vascular disease, and sudden cardiac death [22–26]. Early CRP level elevation in AMI was shown to predict higher risk for future HF development, despite apparent clinical stability [3].

Although CRP level is reported to be associated with infarct size and the extent of myocardial necrosis [2,3] no such difference in LV systolic function was demonstrated between the three CRP groups. Similarly, there were no significant differences between peak creatine phosphokinase and peak troponin. The elevation of LV filling pressure in the presence of preserved cardiac output might suggest greater susceptibility of diastolic events to proinflammatory activation. Early inflammation may lead to acute changes in the Frank–Starling curves of the ischemic stunned heart, resulting in maintained cardiac output and EF at the price of increasing filling pressures. The lack of changes in left atrial volume, a marker of prolonged LV filling pressure elevation, between the groups, further suggests that the changes observed are acute rather than chronic [2,3].

Surprisingly, *E*/*A* ratio was not found to be significantly different between the CRP groups. *E*/*A* is considered a good parameter of LV filling pressure in patients with reduced LVEF. However, it is more challenging in patients with normal or preserved LVEF because the status of myocardial relaxation must be clarified before a reliable assessment of diastolic function and filling pressure can be undertaken. In fact the same *E*/*A* ratio may underlie normal relaxation and LV filling pressure or significant diastolic dysfunction and elevated filling pressure in patients with preserved LVEF. Although our cohort was composed entirely of patients with STEMI, average LVEF was only mildly reduced (48%) and most patients (118, 68%) had a preserved or only mildly reduced LVEF (>45%). The fact that the majority of patients had preserved systolic function precluded estimation of filling pressure just by the *E*/*A* ratio and may have resulted in similar *E*/*A* ratio despite entirely different filling pressures in the CRP groups. In fact, when using the recent recommendations for the evaluation of diastolic function by echocardiography [17] incorporating both *E*/*A*, LA volume, and *E*/*e*′, we found a significant difference in diastolic dysfunction grades between the CRP groups with a majority of patients in the lower tertile having a normal pattern, as opposed to a majority of patients with pseudo-normal

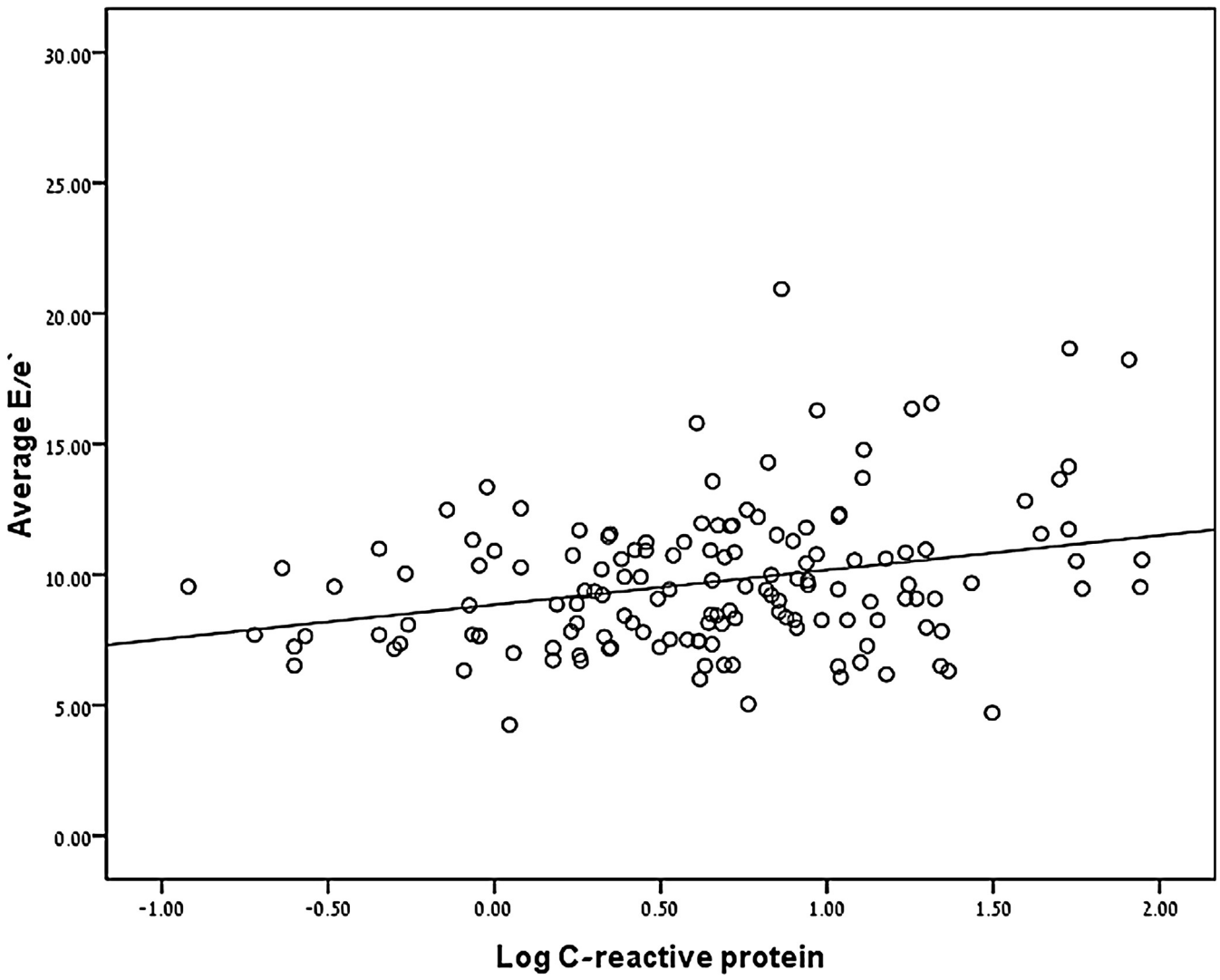

Fig. 2. Correlation between admission C-reactive protein level and E/average e' ratio ($p < 0.001$).

pattern in the upper tertile, suggesting that indeed higher CRP was associated with higher filling pressure despite similar E/A ratio.

E/e' ratio is only a rough estimate of LV diastolic filling pressure, the differences in E/e' ratio among CRP groups were small, and even in the highest CRP group the mean values for E/e' remained below 15 for the septal annulus and below 12 for the lateral annulus which are considered the best cutoff values to diagnose elevated filling pressure [17]. Nevertheless, the prevalence of pathologic E/e' (lateral >12,septal >15), implying elevated left atrial pressure, was higher in the second tertile, and highest in the third tertile of CRP, suggesting that higher CRP is associated with a tendency for pathologic rise in LA pressure. Furthermore, although E/e' is only a rough estimate of LV diastolic filling pressure it remains the most validated non invasive parameter for its estimation, which is a main reason that the joint diastology working group recommends that an evaluation of filling pressure begins with e' [17]. Several studies have shown that the pulmonary capillary wedge pressure is >20 mmHg when E/e' is >15 (e' from the medial annulus) or >12 (e' from lateral annuls) [27–29]. However, although E/e' is rather specific, it is less sensitive for increased filling pressure and many patients with increased filling pressure have an E/e' lower than those values [30].

Thus, we believe that although mean E/e' were below accepted cutoff values [17] for elevated filling pressures in all CRP groups, the small significant difference in its absolute value between groups, and the significant increase in prevalence of pathologic rise in both E/e' septal and E/e' lateral strongly suggests that there was indeed a significant difference in LV filling pressure dependent on CRP levels.

More than half of our patients had an E/e' of 8–15. Recent guidelines advocate the use of other supporting echocardiographic measurements to reinforce LV filling pressure estimation whenever

Table 3
Linear regression model results.

Correlates	E/average e' ratio	
	β	p-Value
Age	1.132	0.259
Gender	2.507	0.013
Diabetes	1.599	0.112
Hypertension	2.247	0.026
Peak troponin	2.085	0.039
LV ejection fraction	0.73	0.466
Anterior infarct	1.131	0. 26
CAD extent	1.185	0.238
Ischemia duration	2.636	0.009
C-reactive protein	2.077	0.04

LV, left ventricular; CAD, coronary artery disease.

E/e' is between 9 and 14 [17]. However, it has been shown that the e' is strongly correlated with invasive measures of myocardial relaxation [31], and that E/e' linearly correlates, throughout all its range with filling pressures in patients with a wide range of ejection fraction [27–29]. Thus, we believe that although mean E/e' were in the indefinite range in most patients, the small significant difference in its absolute value between groups strongly suggests that there was indeed a significant difference in LV filling pressure dependent on CRP levels.

While there were significant differences in E/e' among groups, no significant differences were observed in e' velocity. Although e' velocity correlates well with invasive measures of the time constant of myocardial relaxation (tau) it is not entirely governed by relaxation [32]. The hemodynamic determinants of e' velocity include LV relaxation, preload, systolic function, and LV minimal pressure [17,28]. For preload, although LV filling pressures have a minimal effect on e' in the presence of impaired LV relaxation [17,32], with normal or enhanced basal segmental LV relaxation, as can be encountered in patients with MI involving only the apical segments, preload may influence e' [33,34]. Furthermore, e' is influenced by the type of infarction. It has been shown that e' has lower values in the wall involved with infarction vs the contralateral sites. Therefore, in patients with segmental cardiac disease, absolute individual values of e' may not entirely represent changes in LV relaxation and are also influenced by preload and the segmental nature of disease. Furthermore, an increase in E/e' observed in patients with elevated CRP may represent the combined effects of reduced relaxation or increased preload. Nevertheless, it has been proved that even in patients with segmental systolic dysfunction average e' velocity can be used to correct for the effect of LV relaxation on mitral E velocity, and that the E/e ratio is the best predictor of LV filling pressures, suggesting that irrespective of mechanism, elevated CRP is associated with elevated filling pressure [17,34].

Our findings have several clinical implications. Measuring serum CRP and performing comprehensive diastolic echocardiography during the early stage of AMI could be useful in identifying the patients at increased risk for long-term LV remodeling, even in the presence of preserved LV function. Oduncu et al. demonstrated that long-term pretreatment with statins was associated with lower admission CRP levels in AMI patients, as well as with better myocardial perfusion following PPCI [35]. Past statin treatment had no effect on CRP levels in our cohort, however no information was present regarding their dose and potency. As statins are known to have pleiotropic and anti-inflammatory effects, independent of their lipid-lowering function [36,37], early administration of high-dose potent statins may blunt the acute inflammatory response elicited by an AMI, as indicated in the guidelines [13].

The present study has some limitations that warrant mention. Age-related changes [12] as well as baseline diabetes and hypertension may result in already existing diastolic dysfunction. Bearing these latter possibilities in mind, we excluded patients older than 75 years, whereby the mean age of our cohort was significantly lower than the mean ages of patients in previous trials that assessed diastolic dysfunction in AMI. CRP levels were not consecutively measured throughout hospitalization, thus the magnitude of change and peak serum CRP over time was not determined. Finally, no information was available on long-term outcome, including the development of HF or LV remodeling.

Overall, in our preliminary study, admission CRP levels are associated with some echocardiographic parameters of elevated LV filling pressure in patients with STEMI treated with PPCI. Because of the small differences in E/e' between the CRP groups, and the majority of patients with E/e' ratio between 8 and 15, our data should be interpreted cautiously, and larger clinical trials would be required to prove that CRP levels are associated with elevated filling pressure.

Conflict of interest

None on the part of any author.

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